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SUMMARY CHARACTERIZATIONS OF SELECTED CHEMICALS OF NEAR-TERM INTEREST

APRIL 1976

OFFICE OF TOXIC SUBSTANCES
U.S. ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

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This report includes summary characterizations of 15 chemicals of near-term concern to EPA. The report summarizes (a) health and ecological effects and environmental behavior, (b) sources, environmental levels and exposed populations, (c) technologic and economic aspects and (d) steps that have been taken and are being taken.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Arsenic, Asbestos, Benzene, Benzidine, Cadmium, Ethylene Dibromide, Hexachlorobenzene, Hydrogen Sulfide, Mercury, Platinum, Polybrominated Biphenyls, Polynuclear Aromatic Hydrocarbons, Trichloroethylene, Tris (2,3-dibromopropyl) Phosphate, Vinylidene Chloride		

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Preface

This Report includes summary characterizations of 15 chemicals of near-term interest to EPA. There are many other chemicals of interest, and in the months ahead, similar characterizations of other chemicals will be prepared.

The characterizations are based on information available as of April 1976. As additional information and interpretations of data become available, an updating of the characterizations may be appropriate. To this end, the Office of Toxic Substances would welcome comments on the technical aspects of the Report.

The Report was prepared by the Office of Toxic Substances drawing on information provided by a number of Offices. The Office of Research and Development was particularly helpful in supplying information concerning platinum, hydrogen sulfide, and polynuclear aromatic hydrocarbons.

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ARSENIC

Why Should the Chemical Be of Concern at This Time?

In 1975, OSHA proposed a strict standard for workplace air exposure limits to inorganic arsenic. Earlier EPA sampling had found that atmospheric concentrations near two copper smelters exceeded the proposed limit (Anaconda, Montana; and Tacoma, Washington) and closely approached it at three other smelter sites. Preliminary results of an EPA-sponsored epidemiology study near an arsenical pesticide plant in Baltimore reveal lung cancer rates several times the national average. Congress has proposed that explicit attention be given to establishing an air standard by 1977 in an amendment to the Clean Air Act. A number of arsenical compounds are being considered for rebuttable presumption proceedings under FIFRA/FEPCA.

What Are the Health and Ecological Effects, and Environmental Behavior?

Liver, skin, lung, and lymphatic cancers, and adverse effects on the thyroid gland have been reported in epidemiological studies of occupationally exposed individuals. The main threat of arsenic as a carcinogen is inhalation of the inorganic forms. A preliminary mortality study of the population surrounding Allied Chemical Company's arsenical pesticide plant in Baltimore revealed a lung cancer rate sixteen times the national average. A previous study had shown that retired workers from this plant suffer from lung cancer at a rate seventeen times the national average. A Dow Chemical Company study indicated an excess of lung and lymphatic cancers among their workers who had been exposed to arsenical compounds. Arsenic occurs in two forms: trivalent and pentavalent. Trivalent arsenic is much more toxic than pentavalent, both acutely and chronically. Pentavalent arsenic is often found in metallo-arsenicals, and is of concern because it can degrade into the trivalent form.

A 1972 outbreak of arsenic poisoning in Getchell, Nevada, is attributed to stack effluent from a gold smelter. Studies made abroad have suggested that arsenic may be a skin carcinogen when ingested in drinking water at levels as low as 0.3 mg/l. The debate over the carcinogenicity of arsenic is largely due to the fact that the animal studies conducted to date have not shown a relationship between ingested arsenic and cancer. Organic arsenical compounds may be more hazardous than previously believed. Carbarsone has been reported to produce liver cancer in trout through ingestion (480 mg/100g diet).

Arsenic is particularly toxic to legumes and other crop plants. Depending on the soil type, 6 ppm arsenic can cause a 50 percent growth reduction. Phytotoxic levels of arsenic have been found as far as two miles from the Tacoma smelter. Once combined in soil, arsenic is extremely persistent.

What Are the Sources, Environmental Levels, and Exposed Populations?

Inorganic arsenic is emitted to the air from several sources, including copper, lead, and zinc smelters, glass production plants, coal-burning facilities, cotton gins, arsenical-compound (including pesticides) production plants, and pesticide application. Organic arsenic discharges are associated with the manufacture and use of pesticides. Trivalent arsenic occurs naturally, is a common contaminant of ores, and is the major component of arsenic emissions from smelters. Based on EPA estimates,

the 15 copper smelters contribute most heavily to air emissions of inorganic arsenic. The Anaconda copper smelter in Montana, and the ASARCO copper smelter and arsenic plant in Tacoma, Washington, have been identified as having the highest arsenic emissions. Other industrial sources generally emit less arsenic than copper smelters. Air levels in most urban areas for 1973 and 1974 were at or below the level of detection (0.001 ug/m^3). Levels in areas near smelters ranged from 0.003 to 4.86 ug/m^3.

The land disposal of arsenical wastes can become a long-range public health hazard. A good example is Perham, Minnesota, where eleven people were poisoned by contaminated well water in 1972.

A 1975 survey of drinking water supplies showed that about one percent exceeded the interim drinking water standard of 0.05 mg/l. Trivalent arsenic is found at high levels in some ground water. Underground injection of arsenical pesticide wastes in Philadelphia has contaminated a nearby stream which is being considered for use as a drinking water supply.

Three new technologies for energy production have important arsenic implications. Early data on coal gasification indicate that two-thirds of the arsenic present is volatilized. Oil shale exploitation and geothermal energy development may also release large quantities of arsenic.

What Are the Technologic and Economic Aspects?

In general, particulate control measures (multicyclones, balloon flues, and electrostatic precipitators) are used to reduce arsenic emissions. Baghouses offer the greatest potential for control, but have not been widely adopted by the smelting industry because of high capital and maintenance costs. Costs and feasibility of emission controls will vary from plant to plant. Significant control efforts are being planned at the ASARCO smelter in Tacoma, and are underway at Anaconda Conventional water treatment technology has been shown to be effective in meeting the arsenic drinking water standard. Arsenic concentrations of 0.1 and 1.6 mg/l in wastewater can inhibit waste treatment by activated sludge and anaerobic digestion respectively. Thus, concentrations exceeding these levels can present an additional hazard in waste waters subjected to these treatment methods. Air and water pollution control efforts normally result in a solid waste or sludge. At present, these materials are being stored, pending development of acceptable disposal technologies.

What Steps Have Been Taken, and What Is Being Done?

EPA is locating and monitoring arsenical discharges, and is conducting several studies to determine the toxicity of various arsenical compounds. Limited epidemiological studies are planned to help determine effect levels. Studies have been initiated to determine control technologies and costs for arsenic reduction, and an Air Pollution Assessment Report on Arsenic has been prepared. EPA is considering the development of standards under Section 112 of the Clean Air Act. A review of the use of arsenical pesticides has recently been completed, and research into disposal techniques for arsenical wastes is planned. A Scientific and Technical Assessment Report is planned upon receipt of the NAS study of health effects.

In November 1975, OSHA proposed a workplace exposure limit for inorganic arsenic at 4 ug/m^3 (8 hour, TWA). The previous standard of 500 ug/m^3 for all forms of arsenic would remain in effect only for organic forms.

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ASBESTOS

Why Should the Chemical Be of Concern at This Time?

OSHA has proposed lowering its workplace standard by a factor of ten on the basis of recent epidemiological data suggesting wider spread health effects than previously suspected. A number of major commercial sources of airborne asbestos are limited by EPA regulations. The Agency is investigating taconite and other hard-rock mining operations, where asbestos is a major ore contaminant. There is renewed interest in hazards possibly presented by dust from asbestos brake linings and interior sources. Meanwhile, EPA continues to underscore the hazard of asbestos fibers in water supplies resulting from Reserve Mining Company's operations. EPA's nationwide sampling program is showing levels of asbestos fibers in water supplies, natural runoff, and discharges from manufacturing and mining sites.

What Are the Health and Ecological Effects, and Environmental Behavior?

Airborne asbestos fibers have been known to cause asbestosis, lung cancer, and pleural and peritoneal mesothelioma. OSHA cites a number of studies showing gastrointestinal (GI) cancer in workers exposed to asbestos. In one study of insulation workers in the United States, seven percent of deaths could be attributed to asbestosis, which generally appeared about 20 years after first exposure -- the same latency period as for most cancers. Available epidemiological data show that lung cancer is responsible for as much as 20 percent of all deaths among certain asbestos workers, mesothelioma, 11 percent; and GI cancer, eight percent.

There are few if any data on the dose-response relationships of asbestos fibers in either air or water. Effects of airborne asbestos are far better documented than those of waterborne. OSHA cited workers who had developed mesothelioma at exposure levels below the previous standard of 5 fibers per cubic centimeter in its recent proposal to reduce the level by a factor of ten. There is some evidence that asbestos diseases, including mesothelioma, occur in families of workers exposed to asbestos at levels presumed to be much lower than direct occupational exposure.

Asbestos fibers are extremely resistant to degradation in the environment. Thus far, it has been impossible to demonstrate adverse effects on plants. Some adverse effects on animals have recently been reported.

What Are the Sources, Environmental Levels, and Exposed Populations?

The United States utilized approximately 800,000 tons of asbestos fiber in 1974. Asbestos products are widely used in the construction industry (asbestos-cement pipe, building and other construction products, and floor tile). Other products include friction materials (such as brake linings), felt and paper, packings and gaskets, and fireproof textiles. It has been estimated that 85 percent of the asbestos is tightly bound in products and is therefore not as available to the environment as are airborne and waterborne asbestos fibers generated in the mining and milling of asbestos ore, manufacture and fabrication of asbestos products, and disposal of solid wastes from these processes. Asbestos was used in spray insulation in buildings between 1950 and 1972. This may become a major source of environmental discharge as buildings constructed during this period are demolished.

Asbestos minerals are found throughout the United States. Significant quantities of asbestos fibers appear in rivers and streams draining from areas where asbestos-rock outcroppings are found. Some of these outcroppings are being mined. Asbestos

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fibers have been found in a number of drinking water supplies, but the health implications of ingesting asbestos are not fully documented. Emissions of asbestos fibers into water and air are known to result from mining and processing of some minerals. Asbestiform fibers in the drinking water of Duluth and nearby communities at levels of 12 million fibers per liter have been attributed to the discharge of 67,000 tons of taconite tailings per day into Lake Michigan by Reserve Mining.

Exposure to asbestos fibers may occur throughout urban environments. A recent study of street dust in Washington, D.C., showed approximately 50,000 fibers per gram, much of which appeared to come from brake linings. Autopsies of New York City residents with no known occupational exposure showed 24 of 28 lung samples to contain asbestos fibers, perhaps resulting from asbestos from brake linings and the flaking of sprayed asbestos insulation material.

What Are the Technologic and Economic Aspects?

Coagulation treatment and filtration are necessary to remove contaminant asbestos from water. Filtration technologies for air, while meeting the no-visible-emission standard, permit large quantities of asbestos fibers to escape. Fibrous glass has frequently been substituted for applications requiring insulative properties, but there is some debate over its safety. For some other applications, such as brake linings, economically feasible substitutes may not be available.

There is no inexpensive, standardized analytical method for measuring asbestos, and monitoring costs are very high.

What Steps Have Been Taken, and What Is Being Done?

An air standard has been promulgated for a number of major commercial sources of asbestos fibers. Hard-rock mining and taconite beneficiation, where asbestos is an ore contaminant, are being investigated. Effluent guidelines have been promulgated under the Federal Water Pollution Control Act which, together with the NPDES permit program, should reduce asbestos discharges.

EPA is sponsoring an extensive national asbestos monitoring program. Preliminary findings indicate that asbestos is a widespread contaminant of drinking water. NAS is reviewing the implications of these preliminary findings. EPA's Reserve Mining Task Force is monitoring efforts to halt the discharge of taconite tailings into Lake Superior. Standard analytical methods are being developed for both research and monitoring purposes. A number of epidemiology studies to further clarify the health risks of asbestos are being sponsored by EPA.

In 1972, OSHA established a workplace exposure standard. Last October, OSHA proposed a further reduction in the level. The National Institute of Environmental Health Sciences (NIEHS) is conducting ingestion experiments to clarify health hazards of this route of exposure; EPA is partially sponsoring these studies.

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BENZENE

Why Should the Chemical Be of Concern at This Time?

Benzene, a component in gasoline and an important feedstock for the chemical industry, has been the subject of numerous published reports linking leukemia with worker exposure to it. Large quantities of benzene are discharged into the environment from automobiles, and probably from stationary sources. The Environmental Defense Fund has been particularly concerned about the identification of benzene in drinking water in the parts per billion range. NIOSH has recently recommended a reduction in workplace levels.

What Are the Health and Ecological Effects, and Environmental Behavior?

Numerous fatalities from occupational benzene poisoning have been reported since the early 1900's. After inhalation or ingestion, benzene is absorbed rapidly by the blood. At non-lethal concentrations, a variety of human central nervous system disorders are observed, depending upon the extent of exposure. These include euphoria followed by giddiness, headache, nausea and staggering gait, as well as fatigue, insomnia, dizziness, and unconsciousness. Observed human blood-forming system damage includes anemia, reduction in platelet numbers, and depression of the white blood cell count.

Chronic benzene exposure has also resulted in chromosome aberrations in human lymphocytes. As early as the 1930's, benzene was suspected in cases of leukemia. Available epidemiological data indicate that the compound does induce leukemia, although the data cannot be considered to constitute unequivocal evidence that benzene acting alone is leukemogenic. Attempts by NCI and others to induce leukemia in animals with benzene have not been successful. However, the results of inhalation experiments with mice, the species most susceptible to leukemia, are not yet available.

Based on its physical properties, benzene is expected to be quite mobile and probably persistent. Adverse effects on ecological resources have not been reported.

What Are the Sources, Environmental Levels, and Exposed Populations?

In 1973 over 10 billion pounds of benzene were produced from petroleum and coal in the United States. This volatile, colorless, flammable liquid is used mostly for synthesis of organic chemicals. It has been estimated that at least 80 million pounds of benzene may be lost to the environment during benzene production, storage, and transport, while an upper limit of 650 million pounds may be released during its use to produce other organics. The latter figure was calculated from the difference between 100% yield and the reported yield in these reactions. Therefore, this is only a crude measure of the worst-case benzene emissions during usage. The emissions would be concentrated in the Texas Gulf area and the Northeast.

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It has been calculated that approximately one billion pounds of benzene were released with hydrocarbon emissions from motor vehicles in 1971 in a geographical pattern similar to population distribution. Another 22-24 million pounds of benzene may be released into the environment each year with spilled oil. Hydrocarbon emissions from non-transportation sources, such as coke ovens and power plants, may also contain considerable amounts of benzene. Additionally, benzene is an active ingredient in a number of insecticides and miticides, although the amount of release to the environment from this source has not been calculated.

In an EPA study of organic compounds in the drinking water of 10 cities, benzene was detected in water from four cities at concentrations ranging from 0.1-0.3 ug/l. Previous studies reported levels up to 10 ug/l. Average levels of benzene detected in air in a limited number of studies are in the low ppb range with one high reading of 23 ppm reported in the vicinity of a solvent reclamation plant. No data have been found on levels of benzene in soil, wildlife, and fish. Benzene is widely enough distributed that most people are probably exposed to very low levels; the health implications of this type of exposure are not known.

What Are the Technologic and Economic Aspects?

Reduction in organic compound emissions to achieve the National Ambient Air Quality Standard for oxidants should also result in some reduction in benzene emissions. As a result of lead removal from gasoline, the average content of aromatics, including benzene, in gasoline is likely to increase slowly. However, hydrocarbon emission controls on motor vehicles should result in a net reduction in benzene emissions.

What Steps Have Been Taken, and What Is Being Done?

In 1974 NIOSH published a criteria document for occupational exposure to benzene which recommended adherence to the existing Federal standard of 10 ppm as a time-weighted average with a ceiling of 25 ppm. OSHA is now in the final stages of reviewing its current standard. NIOSH is conducting a retrospective study of benzene mortality and a study of airborne benzene levels in service stations.

EPA has also initiated an air monitoring program which will determine benzene levels in selected areas. Qualitative results obtained to date indicate widespread low-level benzene contamination. Air regulations are not being considered at this time; however, further studies are planned. EPA has conducted a limited survey of drinking water supplies in which benzene was identified in some samples, and has begun a more extensive survey which will seek out benzene as well as a number of other pollutants. EPA has proposed to designate benzene a hazardous substance under section 311 of FWPCA, and ocean dumping is already strictly regulated. A National Academy of Sciences review of the health aspects of benzene being done for EPA should be completed soon. CPSC is also awaiting the results of the NAS study on the health effects of benzene, and will determine if action is appropriate when the results have been received.

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BENZIDINE

Why Should the Chemical Be of Concern at This Time?

Benzidine, a human carcinogen, is used as an intermediate in the manufacture of a number of azo dyes which color textile, leather, and paper products. In addition to the Agency's long-standing concern over liquid effluent discharges containing benzidine, recent research results suggest that some of the benzidine-derived azo dyes may reconvert to benzidine in man, or under certain environmental conditions. The AFL/CIO has expressed strong interest in any action taken on benzidine.

What Are the Health and Ecological Effects, and Environmental Behavior?

For a number of years, the manufacture and use of benzidine have been associated with a high risk of bladder cancer among exposed workers. Many scientists believe that tumors can result from ingestion, inhalation, or skin absorption. A number of animal studies have demonstrated the carcinogenic effects of benzidine. Mice, rats, and hamsters develop liver tumors, and dogs develop bladder cancer. Such studies have many deficiencies for estimating the risk associated with the levels of exposure to carcinogens likely to be encountered in the environment.

Free benzidine has been detected in the urine of monkeys fed benzidine-derived azo dyes, establishing a potential for reconversion of azo dyes to benzidine. Metabolism of benzidine-derived azo dyes may be similar in humans. Japanese silk painters reportedly have a high incidence of bladder cancer, possibly resulting from licking brushes and spatulas coated with benzidine-derived azo dyes. However, the carcinogenicity of such dyes has not been specifically determined.

Industrial data indicate that benzidine entering a waterway dissipates and may be degraded by naturally occurring processes. Confirmatory investigations have not been conducted. Other aspects of environmental behavior have not been addressed. It has been hypothesized that azo dyes can reconvert to benzidine under certain undefined environmental conditions.

What Are the Sources, Environmental Levels, and Exposed Populations?

The three identified manufacturers (Allied, GAF, and Fabricolor) estimate that they produce 45 million pounds of azo dyes annually from benzidine. The dyes are used by about 300 major manufacturers of textile, paper, and leather products. The largest manufacturer (Allied) recently announced its intention to phase out benzidine production.

The principal environmental concern at benzidine production facilities has been the amount of benzidine in the waste effluents discharged to publicly owned waste water treatment works (POTWs). However, the only discharge measurements to date have been made by industry, which has contended that discharges at any facility usually do not exceed one pound per day. Benzidine is believed to be present in the sludge removed from industrial pretreatment plants. The environmental adequacy of land disposal of these sludges is unknown. According to industry data, discharges from the POTW are usually below the limit of detection. However, there are occasionally significant accidental releases to POTWs. Levels of benzidine exceeding 5 mg/l can inhibit anaerobic digestion wastewater treatment processes. Thus, concentrations above this level at the POTW present a problem to POTWs using this process, and a possible hazard to the receiving waters.

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Free benzidine is present in the benzidine-derived azo dyes. According to industry, quality control specifications require that the level not exceed 20 ppm, and in practice the level is usually below 10 ppm. Industry has estimated a total environmental discharge at the 300 user facility sites of 450 pounds per year or about 1.5 pounds per year per facility, assuming all of the free benzidine is discharged in the liquid effluent.

No measurements for benzidine in ambient air, surface water, or drinking water have been reported. Further, no measurements for free benzidine in finished products containing azo dyes have been reported.

What Are the Technologic and Economic Aspects?

The principal liquid effluent control technology currently being used is the reaction of benzidine with nitrous acid. While effective in destroying benzidine, hazardous decomposition by-products may be formed. Industry thus far has rejected carbon adsorption as uneconomical. The costs of treatment at the benzidine manufacturing plants are of far less concern than at the user plants. Thus, there is a continuing industrial emphasis on reducing the levels of free benzidine in dyes, which result from more complete reactions and release less benzidine into the environment.

If limitations were imposed on benzidine production or use, the vacuum would probably be filled by imported benzidine-derived dyes and substitute dyes. However, some of the possible substitutes, such as o-toluidine, are also of environmental and occupational health concern. Industry estimates that adequate substitutes would be three to five times more expensive. In some highly specialized uses, particularly for the halogenated benzidine dyes, a technically adequate substitute may not be available.

What Steps Have Been Taken, and What Is Being Done?

The stringent work place standards required by OSHA because of the carcinogenic nature of benzidine reduce environmental discharges resulting from inadequate housekeeping procedures at benzidine manufacturing sites.

EPA proposed a toxic pollutant effluent standard in December 1973 and is planning to repropose such a standard and a pretreatment requirement during the next few months. The results of current animal experiments at the National Center for Toxicological Research, addressing carcinogenicity and metabolic behavior, should be available within one year. Benzidine is also being examined in the expanded EPA drinking water survey.

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*Synthetic Organic Chemical Manufacturers Association

CADMIUM (Cd)

Why Should the Chemical Be of Concern at This Time?

As evidence that cadmium levels in the environment may be increasing emerges, concern mounts over this substance's ability to accumulate in the body at low level exposures. Cadmium, which is used in a variety of commercial and consumer products, is believed to reach man through a number of routes, particularly as a contaminant of fish and other foods. There is recent concern over the presence of Cd in sludge which might reach the food chain as a result of leaching from disposal sites or its use as a soil conditioner. A proposed amendment to the Clean Air Act calls for explicit EPA attention by 1977 to a possible air standard for cadmium.

What Are the Health and Ecological Effects, and Environmental Behavior?

Cadmium accumulates in the kidney cortex, where it can cause damage to the renal tubules at levels on the order of 200 ppm. The results of autopsy studies show current levels of 15-50 ppm in the kidneys of people over the age of 50 who were not occupationally exposed; the higher levels generally reflect those found in individuals who had been smokers. Autopsy data on the occupationally exposed are inconclusive because samples have been too small.

At high levels of Cd exposure, other effects, such as bone brittling, have been observed, mainly in Japan, where widespread occurrence of Itai-Itai disease caused nearly 100 deaths. These effects resulted from an estimated intake of 600 ug/day. The average American diet contains 50-75 ug/day. Heavy fish eaters receive a higher dose, but well below the levels observed in Japan. About five percent of ingested Cd is retained in the body, and its biological half-life in humans is estimated to be at least 15 years.

Prolonged exposure to cadmium dust can cause emphysema. Recent epidemiological studies indicate abnormally high rates of several forms of cancer due to occupational exposure. Hypertension has been developed in laboratory animals after prolonged exposure to low levels. The presence of Cd in human fetal tissues during prenatal life shows that the metal traverses the placenta. Experimental studies in laboratory animals have confirmed this observation and have also shown that Cd is a potent teratogen.

Cadmium particulate in air falls out into water and soils. Plants take it up from the soil, and people and animals ingest Cd from these sources. Uptake from contaminated water has not been so well documented; it is suspected that this is the significant route of exposure for fish.

What Are the Sources, Environmental Levels, and Exposed Populations?

Cadmium is produced in conjunction with zinc refining. In 1974, the total U.S. consumption of cadmium was about 6300 metric tons, at a cost of about $8500 per metric ton. About one-third was imported. By 1985, demand is expected to reach 9600 metric tons. Of the total use in 1975, about 55 percent went to electroplating, 21 percent to plastic stabilization, 12 percent to pigments, 5 percent to batteries, and 7 percent to a variety of other uses. Major growth is expected in the nickel-cadmium battery industry.

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An EPA-sponsored study estimated that a total of 1800 metric tons of Cd were released to the environment in 1974. Of this, about 20 percent was from zinc mining and smelting, via air, water, and tailings; fifty percent was from such indirect sources as fossil fuel combustion, fertilizer use, and disposal of sewage sludge; and thirty percent was from industrial uses, such as remelting of cadmium-plated scrap, incineration of plastics containing Cd, and electroplating.

The major sources of human exposure are food and tobacco contamination, while direct water and air intake appear to be very minor contributors. Groundwater contamination as a result of waste disposal, however, is common. FDA's marketbasket survey has been identifying low levels of Cd in most composite class samples. Thus, virtually everyone is exposed to trace levels of Cd. Recent studies indicate that, in Sweden, Cd concentrations in wheat may be increasing at a rate roughly proportional to levels of industrial use. Increasing soil levels may result from airborne fallout, fertilizer use, and cadmium in irrigation waters. Cadmium has been identified in soils at several locations at levels of 0.55 to 2.45 ppm.

Cadmium levels of 1 to 10 ug/l have been found in 42 percent of available ambient water samples, with more than 10 ug/l in four percent. Fifty-four percent of the samples did not contain measurable amounts. The annual release of Cd to the air at one copper smelter was estimated to be 250 tons per year. Ambient air levels averaged .031 ug/m^3. Soil levels of Cd were about 1.6 ppm, between one and five miles from the smelter, and were reflected by average findings of 4.7 ppm in leafy vegetables.

What Are the Technologic and Economic Aspects?

Substitutes are or will soon be available for most but not all electroplating uses and for plastic stabilizer use at comparable cost and efficacy.

A Cd level of 0.02 mg/l, has been shown to inhibit wastewater treatment by anaerobic digestion. Should Cd concentrations exceeding that level reach a wastewater treatment plant using anaerobes, a hazard may be presented to the receiving water. Trace contamination of air and water by Cd is common. Removal of such components is usually extremely costly.

What Steps Have Been Taken, and What Is Being Done?

NIOSH is expected to submit a criteria document to OSHA this year, at which time the existing workplace standard will be reviewed. FDA has banned certain uses of Cd pigments and cadmium-containing materials.

Epidemiological studies are being conducted by the World Health Organization to determine whether cadmium may be a factor in hypertension and cardiac disease in humans. NCI is sponsoring studies to investigate the carcinogenic potential of Cd metal, Cd oxide, and Cd sulfide.

EPA has prohibited the ocean dumping of cadmium, except as trace contamination. The effluent guidelines for the electroplating industry address Cd released from this segment of the economy, and hazardous spill regulations include some cadmium compounds among the substances for which spill penalties have been established. An Interim Primary Drinking Water Standard has been issued and pesticides containing Cd are being reviewed for possible Rebuttable Presumption Against Registration proceedings. A Scientific and Technical Assessment Report on Cd has documented health and technological concerns for EPA.

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ETHYLENE DIBROMIDE (EDB)

Why Should the Chemical Be of Concern at This Time?

Ethylene dibromide, which is used primarily as an additive in leaded gasoline, has been identified by the National Cancer Institute (NCI) as an extremely potent and fast-acting carcinogen when administered at high dose levels to animals. Recent monitoring data reveal very low concentrations in air collected from urban and rural areas, and near production and storage facilities. EDF has petitioned EPA to cancel all registered pesticides containing this chemical.

What Are the Health and Ecological Effects, and Environmental Behavior?

EDB is an extremely strong irritant. Chronic exposure can result in liver and kidney damage. High levels of exposure cause immediate depression of the central nervous system, usually resulting in death of laboratory animals. Laboratory animals survive only a few hours of exposure to 200 ppm EDB in air; adverse effects have been noted at exposures as low as 30 ppm. The exposure of domestic animals to EDB has revealed severe reproductive effects. In 1974, NCI issued a memorandum of alert citing preliminary findings of strong carcinogenesis in rats and mice. This document cites a high incidence of squamous cell carcinoma of the stomach in both rats and mice, with tumors observed after only six weeks of exposure. EDB is also mutagenic and teratogenic in animals. In humans, weakness and rapid pulse have been associated with EDB exposure, and, less commonly, cardiac failure leading to death.

The freshwater toxicity of EDB is indicated by the 48-hour Median Tolerance Limit for pan and game fish (15 to 18 ppm). The environmental behavior of EDB is poorly understood. EDB is reportedly short lived in the atmosphere. In soil, EDB is persistent for at least two weeks; however, within a two-month period, it is converted to other compounds. Ethylene, one of these, can reduce yields of fruits, vegetables, and flowering plants.

What Are the Sources, Environmental Levels, and Exposed Populations?

In 1973, approximately 175,000 tons of EDB were produced in the United States. Since sales appear higher than production figures, a small amount of EDB is probably imported. The major producers of EDB are: Houston Chemicals, Ethyl, Dow, PPG, Northwestern, and Great Lakes Chemical. Four of the manufacturing sites are located in Arkansas, one in Texas, and another in Michigan. Eighty to ninety percent of EDB produced in the United States is used as a lead scavenger in gasoline, and it is also registered for use as an insect fumigant and a soil nematocide. The pesticidal use of EDB is less than 1,000 tons per year. EDB is also used as an intermediate in the synthesis of dyes and pharmaceuticals, and as a solvent for resins, gums, and waxes. A small amount of EDB is used in the production of vinyl bromide.

The limited amount of monitoring data available indicates the presence of EDB residues throughout the environment. A Dutch study examining EDB residues in wheat, flour, and bread showed that flour made from wheat treated 13 weeks before analysis contained from 2 - 3 ppm of EDB. Bread made from this flour was found to contain about 0.02 -0.12 ppm of EDB. The fumigation of apples reportedly results in detectable residues on the skin and in the outer pulp for from 4 - 28 days. Low levels of EDB have also been found in the ambient atmosphere of urban and rural areas. Ambient air collected in the vicinity of gasoline stations along highly trafficked arteries in Phoenix, Los Angeles, and Seattle has shown EDB concentrations of 0.07 - 0.11 ug/m^3.

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EDB also escapes into the environment during the production and storage/transfer of gasoline products at oil refineries. Concentrations of EDB in the vicinity of manufacturing plants located in Arkansas ranged from 90 - 115 ug/m^3, while concentrations around a bulk transfer site in Kansas City were 0.2 - 1.7 ug/m^3. Since fully validated monitoring techniques are not yet available, these results should be considered qualitative rather than quantitative indicators.

Although few people live immediately adjacent to production plants where the ambient levels of EDB are the highest thus far recorded, very large populations frequent or live near highly trafficked areas of the type where EDB has been detected.

What Are the Technologic and Economic Aspects?

The elimination of most pesticidal uses for EDB will not result in significant reduction in media exposure, since less than 10 percent of production goes to this use. However, residues in food, which may pose a serious problem, have not been well studied. USDA uses EDB to fumigate certain types of exported and imported grains and produce. Effective substitutes are probably not readily available for some uses, and may be more costly for others. Strict control of EDB emissions from production plants should pose few problems since production is basically a closed system. Major EDB emissions into the environment occur during packaging, and the recycling or capturing of vapors would seem practical. At present, the elimination of EDB from leaded gasoline is not being undertaken by manufacturers. Ethylene dichloride (EDC), which is being used in conjunction with EDB in leaded gasoline could be increased in concentration, and EDB eliminated; however, the environmental impacts of EDC are not clear.

What Steps Have Been Taken, and What Is Being Done?

Actions have been taken to phase out the use of leaded gasoline and are expected to result in a concurrent reduction of exposure to EDB. Gasoline transfer vapor recovery and vehicle evaporative emission controls should further reduce EDB emissions. Monitoring efforts have been initiated to define the zones of impact associated with various stationary and mobile sources of EDB. EPA will gather information on the quantities of EDB, EDC, and other gasoline additives produced, as a result of recently promulgated fuel and fuel additive regulations. Pesticides containing EDB are undergoing special review to determine whether they should be considered candidates for Rebuttable Presumption Against Registration procedures.

OSHA has established a standard for ethylene dibromide to guard against irritation and cumulative hepatic injury; however, the existing standard does not consider the new evidence on carcinogenicity. NIOSH and OSHA are currently reviewing the new evidence to determine whether more rapid action should be taken to revise the standard.

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HEXACHLOROBENZENE (HCB)

Why Should the Chemical Be of Concern at This Time?

Despite recent steps by several States and several companies to reduce environmental discharges of HCB, environmental contamination persists. Recent reports of the occurrence of HCB in human adipose tissues (95 percent of those sampled), the food supply, effluents, drinking water, and pesticides (in addition to registered pesticidal use) add to earlier concerns of EPA, USDA, FDA, and other organizations. In 1973, EPA made a public commitment in response to a petition from USDA to set an HCB food tolerance in 1976.

What Are the Health and Ecological Effects, and Environmental Behavior?

The death of breast-fed infants and an epidemic of skin sores and skin discoloration were associated with accidental consumption of HCB-contaminated seed grain in Turkey in the mid-1950's. Doses were estimated at 50 to 200 mg/day for several months to two years. Clinical manifestations included weight loss, enlargement of the thyroid and lymph nodes, skin photosensitization, and abnormal growth of body hair. HCB levels of up to 23 ppb in blood are believed to have contributed to enzyme disruptions in the population of a small community in southern Louisiana in 1973.

Long-term (up to 3 years) animal ingestion studies show a detectable increase in deaths at 32 ppm, cellular alteration at 1 ppm, biochemical effects at .5 ppm, and behavioral alteration between .5 and 5 ppm. Apparently, the effective dosage to offspring is increased by exposure to the parent. A 12 percent reduction in offspring survival resulted when exposure to very low levels had been continuous for three generations. Teratogenic effects appear minimal.

While HCB appears to have little effect on aquatic organisms, a bioaccumulation factor of 15,000 has been demonstrated in catfish. HCB is toxic to some birds. Eighty ppm caused death, and 5 ppm caused liver enlargement and other effects in quail. The half life of HCB in cattle and sheep is almost 90 days. HCB is very stable. It readily vaporizes from soil into the air; emissions to air in turn contaminate the soil.

What Are the Sources, Environmental Levels, and Exposed Populations?

About 90 percent of the estimated 8 million pounds of HCB produced annually in the United States is as a by-product at 10 perchloroethylene, 5 trichloroethylene, and 11 carbon tetrachloride manufacturing plants. HCB is commonly contained in solid waste and liquid effluents. Most of the remaining production is as a by-product at more than 70 other sites producing chlorine and certain pesticides. About 45,000 pounds per year are released into the environment as a pesticide use. HCB has also been found in the waste tars from vinyl chloride and other chlorine-product plants.

In 1975, forty-six percent of the soil samples collected at 26 locations along a 180-mile transect in Louisiana were contaminated with HCB at levels from 20 to 440 ppb. Parallel sampling of aquatic sediments revealed concentrations of 40 to 850 ppb. Although water samples were generally below 3 ppb, one sample below an industrial discharge contained 90 ppb. Air immediately adjacent to production facilities has shown concentrations from 1.0 to 23.6 ug/m^3. Most of

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the HCB appeared to be associated with particulate, but low levels were found in the gaseous phase as well, which might result from volatization from solid wastes. Samples collected from pastureland near a known HCB production site revealed concentrations in the vegetation from 0.01-630 ppm and in the soil from 0.01-300 ppb.

HCB residues have been found in soil, wildlife, fish,and food samples collected from all over the world. In the United States, HCB residues have been reported in birds and bird eggs collected from Maine to Florida, duck tissue collected from across the country, and fish and fish eggs from the East Coast and Oregon. Animal foods, including chicken feed, fish food, and general laboratory feeds, have been found to contain HCB residues. The frequency of detection of HCB residues in domestic meats has been steadily increasing since 1972, in part because of closer scrutiny. HCB has been detected in trace amounts in only two drinking water supplies.

EPA's monitoring of human adipose tissues collected from across the United States reveals that about 95 percent of the population has trace HCB residues.

What Are the Technologic and Economic Aspects?

If a food tolerance is established by EPA at about .5 ppm (the interim tolerance), there is no reason to believe that substantial numbers of animals or crops will be held off the market. However, a level of .3 ppm or lower would probably prevent the marketing of some products. The feasibility and costs of air emission and water effluent controls, particularly the effectiveness of particulate reduction and of better housekeeping practices, have not been estimated. Effective incineration of wastes has been demonstrated. Proper landfill practice may serve this purpose; however, studies indicate that soil and other covers only delay volatilization.

What Steps Have Been Taken, and What Is Being Done?

In the wake of widespread HCB contamination of cattle in Louisana in 1973, and concern over possible contamination of sheep in California, EPA established an interim tolerance of .5 ppm. Concurrently, the State of Louisiana and several companies took immediate steps to tighten up solid waste practices from manufacturing through disposal. Also, supplies of Dacthal containing 10 percent HCB as an inert ingredient were voluntarily withdrawn from the California market. Several toxicological, monitoring, economics, and related projects were initiated by EPA to provide a better basis for further actions, including the establishment of a tolerance. Also, additional toxicological efforts were undertaken by USDA.

As soon as the needed toxicological data are available, a food tolerance will be established. Also, all pesticide uses of HCB, including pesticides which contain HCB as a contaminant, will be reviewed. Studies of land and ocean disposal methods have been completed. Ocean dumping of HCB-laden tars is prohibited. Although not directly addressed by the NPDES permit program, provisions relating to suspended solids, and oil and grease may provide some degree of control if HCB enters the effluent stream.

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HYDROGEN SULFIDE (H_2S)

Why Should the Chemical Be of Concern at This Time?

Hydrogen sulfide, a colorless gas characterized by its rotten-egg odor, is an emission product from a large number of industrial processes such as kraft paper mills, as well as a naturally occurring chemical. Recently, H_2S has been identified in the exhaust of improperly adjusted catalyst-equipped motor vehicles. This has resulted in considerable public concern because of its strong odor. It has also been identified in emissions from prototype stationary source NO_x reduction catalyst systems. As more and more vehicles become equipped with catalysts, and as the Agency begins to regulate NO_x emissions from stationary sources, H_2S may become a pollutant of greater concern because of its acute toxicity.

What Are the Health and Ecological Effects, and Environmental Behavior?

H_2S is readily absorbed into the blood, with the chief exposure route being inhalation. Test animals exposed to concentrations over 700 ppm exhibited no H_2S in exhaled breath. Systemic poisoning is characterized by respiratory paralysis, occurring when H_2S concentrations in the blood exceed the oxidation capacity. Despite the substance's characteristic odor, which is detectable at levels as low as 0.025 ppm, high concentrations producing toxic effects can be reached almost without warning, because of olfactory fatigue at levels above 50 ppm. Exposures in excess of 400 ppm are considered dangerous, and over 700 ppm, life threatening.

The subacute effects of H_2S exposure are manifested as irritation of the respiratory tract and eyes. Pulmonary edema or pneumonia may result from prolonged exposure to concentrations over 250 ppm. Such exposures over a shorter term may produce temporary symptoms, such as headache, excitment, nausea, dizziness, and painful sensations in the nose, throat, and chest. Chronic exposure to levels of 20 to 30 ppm can lead to conjunctivitis, and 50 to 300 ppm may result in corneal clouding or blurred vision.

What Are the Sources, Environmental Levels, and Exposed Populations?

The primary natural source of H_2S is anaerobic microbial action on organic materials using naturally occurring sulfates. It is also encountered in natural gases and geothermal exhausts, sewers and sewage treatment plants, waters of some natural springs, volcanic gases, and certain mining operations. Because H_2S is soluble in both water and petroleum, it can be transported considerable distances before it is released.

A variety of industrial activities result in the release of H_2S. It is a well-known pollutant at kraft paper mills, oil refineries, coke ovens, natural gas plants, chemical plants, rayon production facilities, rubber production plants, and sugar beet refineries. Fatal and near-fatal H_2S concentrations have been reported at industrial landfills. Environmental levels outside the workplace have not been adequately documented, although there have been a number of reports of odor intensity near plants where reduced sulfur compounds are emitted.

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Recent studies show that H_2S is emitted from maladjusted vehicles with catalytic converters. With adequate oxygen, these catalysts normally convert some of the sulfur dioxide emitted from the system into sulfuric acid; these same catalysts reduce SO_2 into H_2S in the absence of adequate oxygen, usually caused by a malfunctioning air injection pump or a maladjusted carburetor. As more sophisticated catalytic control devices are employed to achieve automotive emission standards, particularly those for NO_x, the potential for H_2S emissions may increase.

Several catalytic reduction processes have been proposed to limit NO_x emissions at stationary sources to achieve the Ambient Air Quality Standard for NO_2. Laboratory tests of prototype systems demonstrate increased production of H_2S. Thus, an increased potential for widespread human exposure to low levels of this substances exists, but the levels cannot be predicted at this time.

What Are the Technologic and Economic Aspects?

Current evidence is that H_2S is emitted in significant quantities from catalyst-equipped automobiles only when such vehicles are defective or maladjusted. Conditions leading to H_2S formation will also result in high levels of carbon monoxide and hydrocarbon emissions. Thus, efforts to ensure proper maintenance of vehicle emission control systems (such as state inspection/maintenance programs) should control automotive H_2S emissions as well, at no additional cost, at least for the 1975/77 model-year vehicles.

Desulfurization of fuels or stack gas feeds could largely eliminate the H_2S problem, but at very high cost and resulting in the generation of large quantities of troublesome sludge.

What Steps Have Been Taken, and What Is Being Done?

H_2S has been, and continues to be, assessed in exhausts from catalyst-equipped automobiles. A fact sheet on current findings has been released to the public. New Source Performance Standards for total reduced sulfur from pulp mills are being developed. These should result in lowered H_2S emissions.

EPA recently contracted with NRC's Committee on Medical and Biologic Effects of Environmental Pollutants to assess the potential environmental problems of H_2S. This study will focus upon effects on man, plants, and animals, and, in addition, will define control technology and areas needing additional research.

OSHA has a health standard of 20 ppm as a ceiling level with a maximum of 50 ppm for 10 minutes once per day only if no other measurable exposure occurs. NIOSH has scheduled H_2S for FY 77 Criteria Document development. The current American Conference of Governmental Industrial Hygienists has recommended an 8-hour TWA of 10 ppm to guard only against conjunctivitis.

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MERCURY (Hg)

Why Should the Chemical Be of Concern at This Time?

Despite recent action by EPA to limit mercury discharges during sludge incineration and through pesticidal use, and earlier Agency efforts to control air emissions and liquid effluent discharges, mercury continues to enter the environment. While more stringent enforcement of existing regulations should be helpful, discharges of mercury from fossil fuel plants, especially those that have shifted to coal from other less contaminated fuels, leaching of mercury from land-disposal sites, particularly into ground water, and urban runoff, are among the currently uncontrolled problems.

What Are the Health and Ecological Effects, and Environmental Behavior?

Hg in many forms is highly toxic to man and other living things. In terms of toxicity, mercury and its compounds can be divided into three categories: 1) alkyl mercury compounds; 2) elemental Hg; 3) inorganic Hg salts and phenyl and methoxy ethyl compounds. Alkyl compounds, particularly methyl mercury, are the most toxic. Over 90 percent of ingested methyl mercury is absorbed in the gastrointestinal tract, and its whole-body biological half life is 70-90 days. Methyl mercury is transported in blood cells to, and concentrates in, brain and other central nervous system tissues where it can cause irreversible damage. In addition, it can cross the placental barrier and cause abnormalities in fetal tissues and irreversible damage to the fetus at levels that appear to cause no symptoms in the mother. Elemental mercury, phenyl and methoxy ethyl compounds, and inorganic mercury salts are far less dangerous than methyl mercury, because less are ingested and the rates of excretion are higher.

The FDA action level of 0.5 ppm of Hg for fish and shellfish, both raw and processed, is based on a 30 ug/day maximum intake of methyl mercury. This is one tenth of the 300 ug/day average intake resulting in a blood level of 0.2 ppb in adults, the lowest level at which neurological symptoms have been observed.

Hg is readily transported to water by leaching from soil and fallout from air; most forms of Hg soil and water can be biologically or chemically transformed to methyl mercury.

What Are the Sources, Environmental Levels, and Exposed Populations?

In 1973, United States use of mercury was slightly less than 1900 metric tons, at an estimated cost of $8800 per metric ton. The chief uses were for battery manufacture (29.9 percent) and chlor-alkali production (24.1 percent). Use in 1965 had been approximately 2700 metric tons; the reduction of use resulted largely from a recognition of the hazards of the substance. In the period 1965 - 1973, several uses (particularly as preservatives and in gold recovery) were eliminated. Hg is also used to make paints and industrial instruments.

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NIOSH estimates that 150,000 workers are exposed to mercury. Because the vapor is colorless and odorless, overexposures can easily go unnoticed until symptoms appear. Of a total of 1900 metric tons used, it has been estimated that as much as 80 percent is discharged into the environment. Distribution of Hg discharges from man-related sources to the environment is about 31 percent to the air, 6 percent to water, and 36 percent to land. Concentrations in the various media are measured in terms of total Hg, rather than the more hazardous methyl forms; thus, the data collected do not represent the true hazard.

Mercury is also a contaminant of coal, and may be a slag runoff problem. In addition, landfills are a source of leaching Hg; this problem may be particularly severe in areas where drinking water supplies are drawn from ground water. Exposure to Hg is widespread, but inadequate documentation of levels of methyl mercury makes estimates of risk difficult.

What Are the Technologic and Economic Aspects?

Because most mercury losses occur in use and disposal of products, recycling may provide the best method for reducing environmental discharges from batteries and instruments. Mercury emissions from the chlor-alkali industry would still be significant, even if state-of-the-art controls are applied to the production stream. Diaphram-cell technology could eliminate mercury emissions, but might add to problems associated with asbestos and lead. New developments in this technology are reducing use of lead and eliminating asbestos; thus increased future reliance on new diaphragm cells may offer the desired reduction in mercury emissions, without the added environmental burden.

What Steps Have Been Taken, and What Is Being Done?

FDA has proposed an action level for Hg in fish and shellfish. As a result of NIOSH recommendations, OSHA is considering revised workplace standards for inorganic and alkyl mercury.

EPA has set a hazardous air pollutant standard for mercury under section 112 of the Clean Air Act, and is considering New Source Performance Standards to require zero emissions of Hg from new chlor-alkali plants.

EPA has addressed the problem through effluent guidelines for a few industrial categories and may expand this coverage in the future. The National Interim Primary Drinking Water Standard for Hg is 2 ppb. Ocean dumping is tightly controlled.

The EPA Administrator recently ordered an end to the registrations of most pesticides containing Hg, and particularly those used in paints, although the decision has been stayed pending completion of judicial review.

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PLATINUM (Pt)

Why Should the Chemical Be of Concern at This Time?

EPA research efforts nearing completion indicate that platinum is more active biologically and toxicologically than previously believed. It methylates in aqueous media, establishing a previously unrecognized biotransformation and distribution mechanism. Because platinum complexes are used as antitumor agents, the potential for carcinogenic activity is present; tests to clarify this aspect should be completed within several months. While low levels of emissions of platinum particulate have been observed from some catalyst-equipped automobiles, the major potential source of Pt is from the disposal of spent catalysts.

What Are the Health and Ecological Effects, and Environmental Behavior?

Prior to EPA's research efforts, the literature cited platinosis as the only known adverse health effect. Platinosis is an allergenic respiratory sensitization to the substance, resulting in a severe asthma-like reaction to low concentrations by sensitized individuals. It is of particular concern to industrial users of Pt, as 50 percent of the workers have shown this syndrome; there is no way to predetermine if individuals are susceptible to it.

Other literature sources indicated that metallic and insoluble forms of Pt were toxicologically inert. Organic complexes of Pt have been used on a limited scale as antitumor treatments, sometimes resulting in toxic effects to the liver.

The health research program has determined the following:

-Metallic Pt and insoluble Pt compounds accumulate in many animal organ tissues, causing various abnormalities.

-Pt methylates in aqueous systems in much the same fashion as does mercury, suggesting a similar, but heretofore unknown, ecosystem transport mechanism for Pt.

-Although Pt is not found in urban or rural air, water, or soil, autopsies have found it in human fat at low levels. It has been hypothesized that this phenomenon may derive from Pt used in dental fillings, thus establishing that Pt may be soluble in human tissue, primarily lipid, and possibly by methylation.

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-Pt in automotive exhaust is principally associated with large particulates coming off the catalyst.

What Are the Sources, Environmental Levels, and Exposed Populations?

Sources of Pt in the United States have been extremely limited, being principally associated with two Pt refining plants and Pt metal fabricators for electrical, chemical, refining, dental, jewelry, and glass industries. Total production and use was about 1.4 million troy ounces (52 tons) in 1971; the projection for 1981 is 2.7 million troy ounces (92.5 tons). Recent introduction of the automotive catalytic converter may result in nationwide exposure to Pt should Pt leach from discarded catalysts. Each catalyst contains 2-5 grams of Pt. Monitoring data to date have revealed virtually no Pt in air, water, or soil.

What Are the Technologic and Economic Aspects?

Should the results of current EPA research efforts to document health hazards of Pt suggest a need to control these exposures, disposal controls would be the most promising, since catalyst disposal is expected to be the largest contributor of Pt to the environment. In addition, the value of the metal would help to offset the cost of reclaiming the Pt from discarded catalysts. If direct vehicular emissions of Pt are found to be significant, particulate traps, which are available at reasonable cost, may provide a technological solution. Other noble metals have been suggested as Pt substitutes; however, even less is known about their potential for hazard.

What Steps Have Been Taken, and What Is Being Done?

An extensive health-effects research program is in progress in EPA, and efforts to characterize platinum in vehicular exhausts are also underway.

OSHA has established a standard for soluble salts of Pt as 0.002 mg/m^3 total Pt, 8-hour TWA. This standard was designed to prevent development of platinosis in workers exposed to airborne Pt.

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POLYBROMINATED BIPHENYLS (PBB's)

Why Should the Chemical Be of Concern at This Time?

In 1973, one to two tons of PBB's, a highly toxic flame retardant, were accidentally mixed into an animal feed supplement and fed to cattle in Michigan. Contamination also resulted from traces of PBB's being discharged into the environment at the manufacturing site and at other facilities involved in handling PBB's. Approximately 250 dairy and 500 cattle farms have been quarantined, tens of thousands of swine and cattle and more than one million chickens have been destroyed, and law suits involving hundreds of millions of dollars have been instituted. Before the nature of the contamination was recognized, many of the contaminated animals had been slaughtered, marketed, and eaten, and eggs and milk of the contaminated animals also consumed. Thus, large numbers of people have been exposed to PBB's. While commercial manufacture and distribution of PBB's have currently ceased, the full extent of the problem has not yet been assessed.

What Are the Health and Ecological Effects, and Environmental Behavior?

Among the 10,000 people who have been identified as having consumed PBB-contaminated meat, milk products, poultry, and eggs, no overt symptoms have been reported to date. Health effects can only be extrapolated from animal data. Based on experimental data, PBB's may be much more toxic than PCB's.

Although no long term toxicity data are available, short term rat, mice, and cattle studies have shown that PBB's may interfere with reproduction and liver functions, promote nervous disorders, and react as a teratogenic agent in tissues. PBB's have produced pathological changes in the livers of rats, mice, guinea pigs, cows, and rabbits. In an experiment with guinea pigs, the chemical was demonstrated to be an immuno-suppressant agent. About 400 cows in herds fed contaminated feed for about 16 days exhibited anorexia, decreased milk production, increased frequency of urination, some lameness, abnormal hoof growth, and shrinking of the udder. Later signs of toxic effects included bloody blebs, malformed or dead fetuses, abscesses, weight loss, and high susceptibility to stress. Non-lactating cows died within six months while the lactating animals survived and gradually improved. Massive liver abscesses were found in dead animals.

Fish taken from streams known to have been contaminated by PBB's have demonstrated that PBB's can bioaccumulate 20,000 to 30,000 times the ambient levels. PBB's are believed to be quite persistent in the environment, with perhaps one-half the lifetime of PCB's. PBB's readily vaporize.

What Are the Sources, Environmental Levels, and Exposed Populations?

PBB's have been used commercially as flame retardant additives in synthetic fibers and molded thermoplastic parts. PBB's have been incorporated into the plastic housings of many commercial products, such as typewriters, calculators, and microfilm readers, and consumer products, such as radio and television parts, thermostats, shavers, and hand tools.

Michigan Chemical Corporation produced approximately 11 million pounds of PBB's from 1970 to 1974. The White Chemical Corporation produced approximately 100,000 pounds of the closely related compounds, octebromobiphenyl and decabromobiphenyl, from

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1970 through 1973. In addition, nine companies have been suppliers of laboratory quantities of PBB's, each producing about five pounds per year. There is no indication of importation of the material.

Monitoring in the Pine River near the facility where PBB's were produced indicated that levels diminished from 3.2 ppb in the ambient stream near the effluent discharge to .01 ppb eight miles downstream. Fish obtained in this eight miles stretch had levels of .09 to 1.33 ppm.

Detailed data are available on PBB levels found in cattle and hogs, with the highest level detected being 2.27 ppm. Data are not yet available on the levels found in any of the 10,000 or more exposed persons.

What Are the Technological and Economic Aspects?

Michigan Chemical Corporation reportedly has paid $20 million in settling a $270 million suit, with claims of $500 million still outstanding. However, the financial dimensions of the incident are still not known.

Among the substitutes for PBB's are the more expensive decabromobiphenyl oxide and several halogenated aliphatic compounds. However, the environmental acceptability of these compounds has not been assessed.

Monitoring methods for PBB's are in the developmental stage. Air monitoring has not yet been attempted.

What Steps Have Been Taken and What Is Being Done?

The State of Michigan has been the focal point for responding to the contamination incident. In addition, USDA, HEW (including FDA, NCI, and CDC), EPA, Michigan State University, and the University of Michigan support a wide array of epidemiological, toxicological, analytical, and related projects to clarify the effects of PBB's on humans and animals and to assess the extent of contamination. The HEW Toxicology Coordinating Committee is preparing a synthesis of available health effects information and will issue a report by June 1975. EPA is providing assistance in monitoring environmental levels of PBB's.

FDA has set temporary action levels for PBB's in contaminated foods and in animal feed. The State of Michigan has issued warnings to sport fishermen along the Pine River.

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POLYNUCLEAR AROMATIC HYDROCARBONS (PNA'S)

Why Should the Chemicals Be of Concern at This Time?

Increased exposure to polynuclear aromatic hydrocarbons (PNA's) and other air pollutants has been implicated by some researchers in increased rates of cancer, especially of the lung. Over 30 PNA's have been identified as urban air pollutants, including several carcinogens. PNA's are emitted during fossil fuel combustion, in natural combustion processes, and as a result of a variety of human activities. A proposed amendment to the Clean Air Act calls for explicit attention by 1977 to a possible air standard for PNA. PNA's have been found at low levels in liquid effluents, some drinking water supplies, and food.

What Are the Health and Ecological Effects, and Environmental Behavior?

Certain PNA's which have been demonstrated as carcinogenic in test animals at relatively high exposure levels are being found in urban air at very low levels. Various environmental fate tests suggest that PNA's are photo-oxidized, and react with oxidants and oxides of sulfur. Because PNA's are adsorbed on particulate matter, chemical half-lives may vary greatly, from a matter of a few hours to several days. One researcher reports that photo-oxidized PNA fractions of air extracts also appear to be carcinogenic. Environmental behavior/fate data have not been developed for the class as a whole.

It has been observed that PNA's are highly soluble in adipose tissue and lipids. Most of the PNA's taken in by mammals are oxidized and the metabolites excreted. Effects of that portion remaining in the body at low levels have not been documented.

Benzo[a]pyrene (BaP), one of the most commonly found and hazardous of the PNA's has been the subject of a variety of toxicological tests, which have been summarized by the International Agency for Research on Cancer. 50-100 ppm administered in the diet for 122-197 days produced stomach tumors in 70 percent of the mice studied. 250 ppm produced tumors in the forestomach of 100 percent of the mice after 30 days. A single oral administration of 100 mg to nine rats produced mammary tumors in eight of them. Skin cancers have been induced in a variety of animals at very low levels, and using a variety of solvents (length of application was not specified). Lung cancer developed in 2 of 21 rats exposed to 10 mg/m^3 BaP and 3.5 ppm SO_2 for 1 hour per/day, five days a week, for more than one year. Five of 21 rats receiving 10 ppm SO_2 for 6 hr/day, in addition to the foregoing dosage, developed similar carcinomas. No carcinomas were noted in rats receiving only SO_2. No animals were exposed only to BaP. Transplacental migration of BaP has been demonstrated in mice. Most other PNA's have not been subjected to such testing.

What Are the Sources, Environmental Levels, and Exposed Population?

PNA's can be formed in any hydrocarbon combustion process and may be released from oil spills. The less efficient the combustion process, the higher the PNA emission factor is likely to be. The major sources are stationary sources, such as heat and power generation, refuse burning, industrial activity, such as coke ovens, and coal refuse heaps. While PNA's can be formed naturally (lightning-ignited forest fires), impact of these sources appears to be minimal. It should be noted, however, that while transportation sources account for only about one percent of emitted PNA's on a national inventory basis, transportation-generated PNA's may approach 50 percent of the urban resident exposures.

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Diesel powered vehicles produce more particulate emissions than gasoline powered; the nature of the fuel is such that the emissions would be expected to contain greater amounts of PNA's, and limited studies have confirmed this. EPA has tested gasoline-powered passenger vehicles to determine the amount of PNA's in the exhaust. However, this characterization is of particulate-associated PNA's; little is known of vaporous components.

PNA's have been detected in urban water supplies at low levels. PNA's in water and soils are adsorbed on minerals or organic particulate matter. Algae and invertebrates contain concentrations as high as 200 times those of the surrounding waters. Levels detected in plants, on the other hand, are slightly lower than soil levels. Sludge samples taken near a steel refinery showed combined benzo[e]- and benzo[a]pyrene levels of 0.91-19.0 mg/kg (dried weight) Liquid effluents did not appear to contain these substances.

Although a variety of PNA's have been observed in particulates from urban air samples, these are not now routinely monitored. Atmospheric concentrations of PNA's are generally represented by measurements of Benzo[a]pyrene (BaP) concentrations. In heavily industrialized areas, BaP levels have been as high as 20 nanograms (ng)/m^3. Urban BaP levels are generally 2-7 ng/m^3; and rural, 0.3 ng/m^3. In 1971-73, nationwide annual emissions of BaP were estimated at 900 tons. It has been estimated that BaP represents 2-5 percent of the total PNA's emitted from automobiles; a similar and as yet undetermined relationship may exist for stationary source emissions.

Because of the large number of sources, most people are exposed to very low levels of PNA's. BaP has been detected in a variety of foods throughout the world. A possible source is mineral oils and petroleum waxes used in food containers and as release agents for food containers. FDA's studies have indicated no health hazard from these sources.

What Are the Technologic and Economic Aspects?

Good particulate emission controls can substantially reduce PNA emissions. However, the costs that would be incurred in further limiting PNA emissions from stationary and vehicular combustion sources are not known. The application of oxidation catalyst exhaust treatment has been effective in dramatically reducing PNA emissions from automobiles when such systems are operating properly. Similar controls at stationary sources may have a similar effect.

What Steps Have Been Taken, and What Is Being Done?

Limitation of carbon monoxide and hydrocarbon emissions from motor vehicles have simultaneously and dramatically reduced PNA emissions. A 1974 analysis of stationary source problems concluded that control regulations designed specifically for BaP or PNA were not warranted or practical, but noted that compliance with existing regulations for incinerators, open burning, coal combustion, and coking operations could significantly reduce PNA emissions. Additional efforts to document stationary source emissions, atmospheric chemistry, and human exposure have been initiated on a limited scale. EPA's STAR document and an NAS report for EPA have detailed much of the hazard and technologic aspects of this class of compounds.

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TRICHLOROETHYLENE

Why Should the Chemical Be of Concern at This Time?

Trichloroethylene (commonly referred to as tri), has been identified by NCI as a carcinogen in laboratory animals. It is widely used for vapor degreasing of fabricated metals and, to a lesser extent, in cleaning fluids. In addition to extensive worker exposure, tri has been detected in ambient air and water in industrial areas, in food, and in human tissues.

What Are the Health and Ecological Effects, and Environmental Behavior?

Tri induces tumors in mice at high dose levels, predominantly liver cancer with some metastases (transfer) to the lungs, according to NCI. Tri is absorbed rapidly by the lungs; only a small amount is eliminated by exhalation, 58-70% being retained. This is slowly eliminated in the urine as trichloroacetic acid (TCA) or trichloroethanol. The first major review of tri poisoning studied 284 cases, including 26 fatalities, in European plants where tri vapors were inhaled. Results indicated that toxic action involves the central nervous system. A number of short-term studies indicate that exposure to a concentration of 100 ppm in air may interfere with psychophysiological efficiency. In one study, six students exposed to 110 ppm for two four-hour periods separated by 1-1/2 hours showed significantly lower levels of performance in perception, memory, and manual dexterity tests. A confirmatory test using six tri workers produced almost identical results. There is a reported case of a man operating a metal degreaser who lost his sense of taste after one month's exposure to concentrations of tri which "occasionally escaped in sufficient quantities to be visible". Two months later he lost facial mobility and sensation, and developed EEG changes which did not clear up during the following two years.

Tri has been frequently detected in the environment; however, the behavior and transport have not been documented. Adverse ecological effects have not been reported.

Because of its low solubility, high vapor pressure, and high photodegradation rate at sea level (half life in air is about eight hours), tri is not expected to accumulate in the atmosphere. Its half life in water is on the order of months.

What Are the Sources, Environmental Levels, and Exposed Population?

Domestic tri production in 1974 was about 215,000 tons by five producers. Over 90% is used for vapor degreasing of fabricated metals. Ambient concentrations in the atmosphere of industrialized areas have been estimated by industry to be 2-16 ppt. Water concentrations are about 0.1 ppt. The character of the water was not defined, but trace amounts of tri have been identified in drinking water by EPA.

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Over 200,000 workers are exposed to tri. The general public is exposed via inhalation of cleaning fluids and ingestion of foods, spices, and medicines from which undesirable components have been removed by extraction with tri. Residues ranging from 0.02 to 22 ppt have been detected in foodstuffs, and concentrations of up to 32 ppt have been detected in human tissue.

There is only one reference to degreasing equipment which concerns control of vapor emissions. In those cases where both an exhaust system and a degreaser vapor condensation system were in use, the average concentration of tri in air near the operation dropped from 105 ppm to 30 ppm.

What Are the Technologic and Economic Aspects?

Both methyl chloroform and perchloroethylene should be considered as possible substitutes for tri as degreasing agents. Both appear less damaging to air quality. However, methyl chloroform may adversely impact the ozone layer. All three are comparably priced and, since many users have already made this change, the economic impact should be minor. Closed loop systems could permit recovery of tri; however, this may represent a higher cost factor than use of substitutes. Preliminary NCI studies indicate that perchloroethylene may also present health hazards.

What Steps Have Been Taken, and What Is Being Done?

In October 1975, OSHA proposed a reduction in the workplace standard, and is currently reviewing the proposal, together with possible standards for methyl chloroform and perchloroethylene. CPSC is preparing a monograph on consumer exposures and possible hazards.

Tri producers are undertaking epidemiological studies, long-term animal studies, long-term animal inhalation studies, and an in-depth literature survey which have recently been started for the Manufacturing Chemists Association.

Since trichloroethylene contributes to photochemical smog, State Implementation Plans provide a mechanism for limiting emissions. Detailed health, environmental, and economic analyses are planned. NPDES permits limiting BOD_5, COD, and suspended solids also provide some control over effluent discharges of tri.

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TRIS (2,3-DIBROMOPROPYL) PHOSPHATE (TBPP)

Why Should the Chemical Be of Concern at This Time?

TBPP is presently the most popular flame retardant additive for acetate and polyester fabrics which are widely used in children's sleepwear. Recent EPA-funded experiments have shown that TBPP is a mutagen in a microbial assay that is also being considered for use as a screen for carcinogenic potential. Crude experiments have suggested that TBPP is present in the waste water from home laundry of such sleepwear. The Environmental Defense Fund recently requested the Consumer Product Safety Commission (CPSC) to take steps to reduce the hazards associated with TBPP and Ralph Nader has asked the Senate Commerce Committee to hold hearings. Both CBS and NBC have raised the issue of continuing use of TBPP in their National news coverage.

What Are the Health and Ecological Effects, and Environmental Behavior?

TBPP is mutagenic in the "Ames bioassay" which is regarded by some toxicologists as a screen for carcinogens. This has been confirmed by two independent investigators.

In feeding experiments, dose-related accumulations of TBPP, or its metabolites, were found in rats given 100 to 1000 ppm for 28 days. Six weeks after withdrawal, no residues were found and no histopathology was detected. A degradation product (2,3-dibromopropanol) was found in the urine of rats after oral administration or dermal exposure, indicating that TBPP reaches a number of organs.

Laboratory experiments have shown that aqueous extracts of fabrics containing TBPP at extremely low levels are lethal to fish even after the fabric had been laundered. Erratic behavior, possibly resulting from central nervous system involvement, was noted before death. Full confirmation of these findings has not yet been performed. Environmental biodegradation occurs, but the rate is not known.

TBPP is also a mild sensitizing agent in humans. However, no allergic responses have been reported from consumer or occupational exposure.

What Are the Sources, Environmental Levels, and Exposed Populations?

About 65% of the 10 million pounds of TBPP produced annually in the United States by six manufacturers are applied to fabrics used for children's clothing, with the remainder used as a flame retardant in other materials, such as urethane foams. A significant portion of the total, perhaps ten percent, reaches the environment from textile finishing plants and laundries. Most of the rest will eventually find its way into solid wastes (manufacturing waste and used clothing). Environmental levels of TBPP near manufacturing plants, dumps, mills, and laundries have not been measured.

TBPP is added to fabrics used for children's garments to the extent of 5-10 percent by weight. A child wearing such a garment and chewing on a sleeve or collar could easily ingest some TBPP, particularly if the

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garment had not been laundered before use. The effects of saliva, urine, or feces on the extractability of TBPP or on its absorption through the skin have not been measured.

What Are the Technologic and Economic Aspects?

At current production levels, TBPP gross sales are about 10 million dollars per year. Substitute materials or methods for meeting the flammability requirements for children's sleepwear are probably available. However, quantitative data on the cost, performance, and safety of substitute materials and methods are not available. The major costs would be in the product development and application areas.

What Steps Have Been Taken, and What Is Being Done?

The mutagenicity of TBPP was originally discovered in a small EPA screening program. The limited available data on the environmental effects of TBPP have been compiled and transmitted to the Consumer Product Safety Commission, the National Institute for Occupational Safety and Health, the Toxicology Coordinating Committee of the Department of Health, Education, and Welfare, the NAS committee on the Fire Safety Aspects of Polymeric Materials, and several other groups concerned with flame retardants, including some TBPP manufacturers and users. The National Cancer Institute is currently conducting carcinogenicity tests on TBPP in rats and mice; results are expected in early 1977.

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Why Should the Chemical Be of Concern at This Time?

Vinylidene chloride (VDC), an important monomer in the manufacture of methyl chloroform and of Saran and other plastics, is of particular concern because the manner in which the problem is emerging is similar to earlier developments concerning vinyl chloride. In January 1976, NIOSH reported that about 60 percent of examined workers in a New Jersey plant using VDC had developed liver disorders, and announced its intention to follow up. Previous laboratory animal studies had suggested that VDC might be a liver carcinogen, as well as produce a number of other adverse health effects. A substantial amount of VDC is vented to the atmosphere during production, polymerization, and fabrication.

What Are the Health and Ecological Effects, and Environmental Behavior?

Vinylidene chloride has recently been reported to cause liver impairment. Twenty-seven of forty-six workers examined at the BASF Wyandotte VDC polymerization plant in South Kearny, New Jersey, showed 50 percent or greater loss in liver function. Other examinations indicate that VDC is biochemically altered in the body and may form intermediates similar to the cancer-producing metabolites of vinyl chloride.

Inhaled VDC is reported to produce liver tumors in rats at 200 ppm. Inhalation experiments with animals showed that VDC causes liver and kidney damage. When rats were pre-exposed to vinyl chloride and then tested with VDC, the acute toxicity of VDC was greatly enhanced. Concurrent exposure reduces the acute effects and may potentiate the carcinogenic effects. This is important because a significant part of polymer production involves the use of both chemicals.

As yet, the ecological effects and environmental behavior of VDC in either air or water have not been studied. Its highly reactive nature would seem to support a thesis that it is relatively short-lived in air, possibly on the order of several hours.

What Are the Sources, Environmental Levels, and Exposed Populations?

Dow Chemical and PPG Industries annually produce 270 million pounds of VDC monomer in three Gulf Coast plants. About 50 percent is used in the production of methyl chloroform by PPG. The remainder is polymerized to plastic resins at 12 facilities owned by a number of companies throughout the country. The resin is then fabricated into plastics at 60 to 75 plants. It has been estimated that about four million pounds of VDC were lost to the air in 1974. One EPA-funded report estimates that as much as 25 percent of the VDC used in any given Saran production run is disposed of in landfill, primarily in polymerized form, but there are no estimates of the levels of unreacted monomer.

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In the past, worker exposure has generally not been monitored. Tests demonstrate that 20,000 ppm can easily be attained in the immediate vicinity of a spill. In some cases, past worker exposures to VDC may have exceeded those to vinyl chloride (which were measured at 300-1000 ppm before OSHA limits were imposed). The odor threshold of VDC is 500 ppm.

What Are the Technologic and Economic Aspects?

The primary requirement for reduction of exposure to VDC would be to limit emissions through improved housekeeping procedures in the industry. The type of control technology used to control vinyl chloride should be applicable to VDC production.

What Steps Have Been Taken, and What Is Being Done?

The American Conference of Governmental Industrial Hygienists has established a threshold limit value of 10 ppm.

NIOSH is planning to monitor the follow-up studies on workers at the South Kearny BASF plant and will survey other VDC production sites in 1977 to determine if a workplace standard should be recommended.

EPA is preparing an assessment of the air pollution problems associated with VDC production and use. Fetotoxicity and embryotoxicity have been demonstrated under EPA-funded contracts. Data on environmental effects of VDC are also being obtained.

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